Application No. 09/912,670
Filed: July 23, 2001
TC Art Unit: 1644
Confirmation No.: 6394

REMARKS

The Applicants have filed a Request for Continued Examination in the above-identified application (May 11, 2005). Claim 24 was previously rejected as obvious over U.S. Pat. No. 6,048,850 in view of Lipton et al. Claim 43 was rejected as obvious over U.S. Pat. No. 6,048,850 in view of Lipton et al. and further in view of Singh et al. Claims 42 and 43 were rejected as not supported in the written description. In the Preliminary Amendment filed simultaneously with the Request for Continued Examination, these rejections were respectfully traversed in view of the accompanying remarks and reconsideration was requested. The Applicants now submit Supplemental Remarks and an Inventor's Declaration showing practice of the method of the invention.

The Applicants submit that the obviousness rejection of the pending claims has been overcome because there was no motivation to combine the references cited by the Examiner without impermissible hindsight reference to the Applicants' claims. US Patent No. 6,048,850 of Young et al. relates to methods of modulating prostaglandin H synthase-2 (PGHS-2) gene expression or the activity of the PGHS-2 gene product. There is no motivation in the teachings of this patent to look to Lipton et al. as Young et al. is not concerned with inflammation in general but, more specifically, with achieving a reduction in prostaglandins in inflamed cells without altering prostaglandin production in other cells, as taught at col. 1, lines 59-61. Furthermore, all that Young et al. teaches, including at the place cited by the Examiner, is that the compounds of *that* invention may be administered by *any* means (*see*, col. 32), including being targeted to specific sites of inflammation. The Young et al. patent teaches nothing concerning how other anti-inflammatory compounds that would function via a different mechanism might be administered. The suggestion to look to Lipton et al. for use of α-MSH has to have come from the Applicants' claims.

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Lipton et al., which relates to the anti-inflammatory actions, specifically, of α-MSH as a representative neuroimmunomodulator, teaches, e.g., the "central and/or peripheral administration of α-MSH . . . within the brain tissue" (p. 141, 1st col., last paragraph) and "marshalling the anti-inflammatory influences of the peptide through stimulation of both CNS and peripheral melanocortin receptors" (p. 144, 2nd col., last paragraph). There is no motivation in the teaching of this reference to look to Young et al. as that reference teaches a method of reducing prostaglandins in inflamed cells without altering prostaglandin production in other cells, and reducing the presence of prostaglandins is of no interest to Lipton et al.

Without impermissible hindsight, there is no hint in either Young et al. or Lipton et al. that one of ordinary skill who was interested in either of these two different classes of compounds could learn anything about the appropriate routes of administration of that compound by looking to the other reference. And, as shown by the experimental results presented in the Taylor Declaration, only a localized injection of genetic material coding for alpha-MSH as particularly claimed, and therefore a localized production of alpha-MSH in the eye, was effective in suppressing the severity and duration of autoimmune disease in the eye. Thus, contrary to the teachings of Young et al. with respect to reducing prostaglandins in inflamed cells, when the method of treatment is to administer alpha-MSH to control inflammation, only localized injection produces useful results.

Applicants submit that all claims are in condition for allowance and such action is requested.

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The Examiner is encouraged to telephone the undersigned attorney to discuss any matter that would expedite allowance of the present application.

Respectfully submitted,

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